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FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH, LIFESCI' ENTERED AT 10:05:49 ON 27 JUL 2009

- L1 403075 S (TRANSFER? OR TRANSPORT?) (6A) (TETANUS(W) TOXIN OR PROTEIN OR P
- L2 1242 S (NEURAL OR NEURONAL) (5A) (TRANSFER? OR TRANSPORT?) (6A) (TETANUS
- L3 44815 S BDNF OR NT-4 OR GDNF
- L4 15 S L2 AND L3
- L5 11 DUP REM L4 (4 DUPLICATES REMOVED)
- => d au ti so pi 1-11 15
- L5 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
- AU Paterson, David S.; Thompson, Eric G.; Belliveau, Richard A.; Antalffy, Bobbie A.; Trachtenberg, Felicia L.; Armstrong, Dawna D.; Kinney, Hannah C.
- TI Serotonin transporter abnormality in the dorsal motor nucleus of the vagus in Rett syndrome: Potential implications for clinical autonomic dysfunction
- SO Journal of Neuropathology & Experimental Neurology (2005), 64(11), 1018-1027

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- AU Kirihara, Kenji; Suga, Motomu; Tochigi, Mamoru; Araki, Tsuyoshi; Yamasue, Hidenori; Kasai, Kiyoto; Sasaki, Tsukasa
- TI Psychophysiological and neural image-based endophenotype in schizophrenia
- SO Bunshi Seishin Igaku (2005), 5(2), 113-125 CODEN: BSIUAZ; ISSN: 1345-9082
- L5 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
- IN Roux, Sylvie; Brulet, Philippe; Saint, Cloment Cecile; Barbier, Julien; Molgo, Jordi
- TI Construction of fusion protein of GFP-TTC (tetanus toxin C fragment) and uses for in vivo modulation of neuronal transport
- SO U.S. Pat. Appl. Publ., 39 pp., Cont.-in-part of U.S. Ser. No. 816,467. CODEN: USXXCO

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	WO	2005	0255	92		A2		2005	0324		WO 2	004-	EP10	991		2	0040	915	
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- AU Dezawa, Mari; Kanno, Hiroshi; Hoshino, Mikio; Cho, Hirotomi; Matsumoto, Naoya; Itokazu, Yutaka; Tajima, Nobuyoshi; Yamada, Hitoshi; Sawada, Hajime; Ishikawa, Hiroto; Mimura, Toshirou; Kitada, Masaaki; Suzuki, Yoshihisa; Ide, Chizuka
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- SO Handbook of Experimental Pharmacology (2004), 157(Antidepressants), 3-33 CODEN: HEPHD2; ISSN: 0171-2004
- L5 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
- IN Tuszynski, Mark H.
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- TI GDNF pre-treatment aggravates neuronal cell loss in oxygen-glucose deprived hippocampal slice cultures: a possible effect of glutamate transporter up-regulation
- SO Neurochemistry International (2003), 43(4-5), 381-388 CODEN: NEUIDS; ISSN: 0197-0186
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- TI Increased expression of brain-derived neurotrophic factor induces formation of basal dendrites and axonal branching in dentate granule cells in hippocampal explant cultures.
- SO The Journal of neuroscience: the official journal of the Society for Neuroscience, (2002 Nov 15) Vol. 22, No. 22, pp. 9754-63.

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- TI Up-regulation of the neuronal serotoninergic phenotype in vitro: BDNF and cAMP share Trk B-dependent mechanisms
- SO Journal of Neurochemistry (2002), 83(6), 1525-1528 CODEN: JONRA9; ISSN: 0022-3042
- L5 ANSWER 11 OF 11 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
- AU Perrelet, D. [Reprint author]; Sagot, Y.; MacKenzie, A.; Smith, G. M.; Kato, A. C.
- TI Adenoviral gene transfer of CNTF and NAIP can rescue motoneurons after neonatal sciatic nerve axotomy.
- SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 490. print.

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 - 1. Los Angeles, California, USA. November 7-12, 1998. Society for Neuroscience. ISSN: 0190-5295.

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- L5 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
- AB A protocol for use of growth factors to stimulate neuronal cell growth and activity in trkB receptor containing cortical tissues, including the entorhinal and hippocampal cortices. The method introduces exogenous growth factor, such as BDNF, NT-4/5 and NT-3, into the EC. The method is useful in therapy of defective, diseased and damaged neurons in the mammalian brain, of particular usefulness for treatment of neurodegenerative conditions such as Alzheimer's disease or for normal aging. A microarray anal. is presented in the examples to demonstrate the gene expression changes following infusion of BDNF in the rat brain.
- L5 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN
- AB Besides its neurotrophic and neuroprotective effects on dopaminergic neurons and spinal motoneurons, glial cell line-derived neurotrophic factor (GDNF) has potent neuroprotective effects in cerebral ischemia. The protective effect has so far been related to reduced

activation of N-methyl-d-aspartate receptors (NMDAr). This study tested the effects of GDNF on glutamate transporter expression, with the hypothesis that modulation of glutamate transporter activity would affect the outcome of cerebral ischemia. Organotypic hippocampal slice cultures, derived from 1-wk-old rats, were treated with 100 ng/mL GDNF for either 2 or 5 days, followed by Western blot anal. of NMDAr subunit 1 (NR1) and two glutamate transporter subtypes, GLAST and GLT-1. After 5-day exposure to GDNF, expression of GLAST and GLT-1 was up-regulated to 169 and 181% of control values, resp., whereas NR1 was down-regulated to 64% of control. However, despite these changes that potentially would support neuronal resistance to excitotoxicity, the long-term treatment with GDNF was found to aggravate the neuronal damage induced by oxygen-glucose deprivation (OGD). increased cell death, assessed by propidium iodide (PI) uptake, occurred not only among the most susceptible CA1 pyramidal cells, but also in CA3 and fascia dentata. Given that glutamate transporters are able to release glutamate by reversed action during energy failure, it is suggested that the observed increase in OGD-induced cell death in the GDNF -pretreated cultures was caused by the build-up of excitotoxic concns. of extracellular glutamate released through the glutamate transporters, which were up-regulated by GDNF. Although the extent and consequences of glutamate release via reversal of GLAST and GLT-1 transporters seem to vary in different energy failure models, the present findings should be taken into account in clin. trials of GDNF.

- ANSWER 9 OF 11 MEDLINE on STN DUPLICATE 1 L5During limbic epileptogenesis in vivo the dentate granule cells (DGCs) AB exhibit increased expression of brain-derived neurotrophic factor (BDNF), followed by striking morphologic plasticities, namely the formation of basal dendrites and the sprouting of mossy fibers. We hypothesized that increased expression of BDNF intrinsic to DGCs is sufficient to induce these plasticities. To test this hypothesis, we transfected DGCs in rat hippocampal slice cultures with BDNF or nerve growth factor (NGF) via particle-mediated gene transfer, and we visualized the neuronal processes with cotransfected green fluorescent protein. Transfection with BDNF produced significant increases in axonal branch and basal dendrite number relative to NGF or empty vector controls. Structural changes were prevented by the tyrosine kinase inhibitor K252a. Thus increased expression of BDNF within DGCs is sufficient to induce these morphological plasticities, which may represent one mechanism by which BDNF promotes limbic epileptogenesis.
- ANSWER 10 OF 11 CAPLUS COPYRIGHT 2009 ACS on STN L_5 The effects of brain-derived neurotrophic factor (BDNF) and cAMP AΒ on the neuronal serotoninergic phenotype were studied in primary cultures of E14 rat embryonic rostral raphe. Short treatments (for 18 h) with BDNF or dibutyryl-cAMP induced an almost two-fold increase in the number of serotoninergic neurons and a dramatic extension and ramification of their neurites. These changes were associated with marked increases in the levels of mRNAs encoding the serotonin transporter, the 5-HT1A and 5-HT1B receptors and the BDNF receptor tyrosine kinase B (TrkB). Concomitant blockade of tyrosine kinases by genistein suppressed all the upregulating effects of BDNF and cAMP on 5-hydroxytryptamine (5-HT) neurons. These findings suggest that an auto-amplifying mechanism underlies the promoting effect of BDNF on the differentiation of serotoninergic neurons through TrkB activation, which is also triggered by cAMP.
- L5 ANSWER 11 OF 11 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN